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CONTENTS

A Survey of Sudden Deaths at Children's Hospital from 1943 to 1948. *William Orr, M.D., Warren Preisser, M.D., and John E. Cassidy, M.D.* 55

Dentigerous Cyst. *Bennett Olshaker, M.D.* 64

Congenital Urethral Stricture. *William M. Crowell, M.D., and Robert H. Anderson, M.D.* 68

Atropine Poisoning. *William Sager, M.D.* 74

Clinico-Pathological Conference. *William M. Crowell, M.D., John A. Washington, M.D., and E. Clarence Rice, M.D.* 78

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A SURVEY OF SUDDEN DEATHS AT CHILDREN'S HOSPITAL FROM 1943 TO 1948

SPECIAL REPORT

William Orr, M.D.
Warren Preisser, M.D.
John E. Cassidy, M.D.

This paper is a statistical review of sudden deaths through the years 1943 to 1948 inclusive, a six year period. During this time there were 1,345 deaths of which we have been able to obtain 1,241 charts. We feel that this number of charts is representative of the period. Each chart was carefully reviewed to determine the duration of illness, period of hospitalization, and cause of death. We have considered all cases dying in less than twenty-four hours after the onset of the illness as sudden death, and in a few cases with an explosive onset, we have extended the time to thirty hours as will be indicated when those cases appear in the discussion. No attempt was made by the authors to interpret duration of illness or diagnosis. The material is presented as obtained from the history and records, for example, a case of diarrhea and marked dehydration dying in twenty hours after onset of illness was considered as having a twenty hours' duration though the history may not have been reliable. We have included cases of idiopathic cardiac hypertrophy with dilatation and congestive heart failure under the category of congestive heart disease but these will be noted in the year to year discussion. In considering whether a cause of death was proven we have relied on autopsies, diagnostic laboratory findings, x-rays, and direct observation, for instance, larnygoscopy or surgery. In the year 1943 there were 279 deaths and 37 sudden deaths or 13.6 per cent of all deaths were sudden deaths.

Of these 37, 9 were in prematures and 7 of the 9 had autopsies. Six of those autopsied had atelectasis. All 9 are considered proven because of the history and size of the infant. Of these, 5 were under two and one-half pounds, 3 were between two and one-half pounds and four pounds, and 1 was four pounds, seven ounces. The average hospitalization was eight hours with one dead on arrival and two living twenty-two hours.

There were four deaths due to Waterhouse Friederichson Syndrome or 10.5 per cent of the sudden deaths. All four had autopsies and were proven. The youngest was a seven week old white male and the oldest, a ten year old white male. The average hospitalization was seven hours. One of the patients lived twenty-two hours in the hospital. He had felt drowsy for six hours prior to admission but has been included in the sudden death group.

There were four cases of penumonia or 10.5 per cent of sudden deaths, all of whom had autopsies. Three of the cases had an average illness of sixteen hours. The other patient (a three month old white male) has been included because the child who had been hospitalized for six days with pneumonia was discharged only to be found dead in bed by a nurse. The autopsy revealed pneumonia, atelectasis, and tracheo-bronchitis.

There were two deaths due to congenital heart diseases or 5.2 per cent, one of whom had a post mortem examination showing multiple cardiac anomalies and congestive heart failure. The other had a murmur and was cyanotic but no autopsy was done.

There were two deaths due to meningitis: one was H. influenzal in type who had a positive spinal fluid culture, while the other was proven to be due to the pneumococcus by post mortem examination and spinal fluid culture.

Under other causes we have included those cases which accounted for only one or two deaths each year. One was an eight year old colored male hemophiliac with multiple internal hemorrhages as proven by autopsy.

Two deaths were due to malnutrition and dehydration. One was a nineteen month old colored male who had no autopsy; and the other, a three month old white male whose post mortem examination showed marked malnutrition and dehydration with atelectasis and pulmonary congestion and edema. One stillborn is included for the sake of completeness since it was given a hospital number and an autopsy was performed showing atelectasis and cardiac hypertrophy.

There was one six month colored female who died of intussusception and shock proven by autopsy.

One case of acute congestive heart failure due to paroxysmal tachycardia and one case of supra laryngeal edema were reported. The latter had no autopsy but did have an ear, nose, and throat consultation proving the diagnosis.

There was one case of septicemia due to a gram negative bacteria not further identified.

There were two deaths listed as having occurred by unknown causes. One was a two and one-half month white female who was dead on arrival. The autopsy showed thymic hyperplasia, bilateral cystic ovaries, and congested choroid plexus. The child had been sick for seven hours. She was allergic to milk but was doing well on a high protein formula. She was given phenobarbitol one-eighth grain at 2:00 A.M. and was found later that morning to be lying on her stomach not breathing. The other patient was a nine year old white female who was in the hospital for five minutes. Autopsy revealed acute splenitis, cystic ovaries, hepatomegaly, acute enteritis, and pulmonary congestion. She had been ill for twelve hours with headache, arthralgia, and petechial hemorrhages in skin.

The operative deaths will be discussed later as a group.

Cause	*No.*	*% SD*	*Aut.*	*Pr.*
Premature	9	23.7	7	9
Wat. Fr. Syn.	4	10.5	4	4
Pneumonia	4	10.5	4	4
Cong. Ht. D.	2	5.2	1	1
Meningitis	2	5.2	1	2
Other	8	21.0	6	7
Unknown	2	5.2	2	?
Operative	6	15.7	6	6

In 1944 there were 266 deaths and 37 sudden deaths. There were 5 deaths due to prematurity, 4 of whom had autopsies and showed atelectasis. All of these were under three and one-half pounds.

All 5 deaths due to Waterhouse Friederichson Syndrome had autopsies. Four cases of laryngo-tracheo bronchitis were reported, three proven by autopsy.

There were four cases of asphyxia, one proven by bronchoscopy and due to aspiration of a bean. One case was due to strangulation with bed clothes, and two cases who had autopsies were due to aspiration of gastric contents, one because of pyloric stenosis.

Three cases of trauma all with fractured skulls and laceration of the brain occurred this year.

Three children died of severe burns; none had autopsies. There were two cases diagnosed as congenital heart disease, one proven by post mortem examination

Under the other causes are medulloblastoma (in a 12 years old white male), a miliary tuberculosis (in a 20 months white female), a hypoprothrombinemia with intracranial hemorrhage (in 6 weeks old white female), a massive lobar pneumonia (in a 1 month old white female), a diarrhea and dehydration (in a 9 months old white female), a staphylococcus septicemia (in a 7 months white male), and an influenzal meningitis (in a 4 months old white female) all proven by post mortem examination.

Cause	*No.*	*% SD*	*Aut.*	*Pr.*
Premature	5	13.4	4	5
Wat. Fr. Syn.	5	13.4	5	5
L. Tr. Br.	4	10.5	3	3
Asphyxia	4	10.5	2	4
Trauma	3	8.0	2	3
Burns	3	8.0	0	3
Cong. Ht. D.	2	5.2	1	1
Other	7	18.4	7	7
Operative	4	10.5	4	4

In 1945 there were 256 deaths and 30 sudden deaths. There were 6 cases of meningitis, 2 due to H. influenza, 2 due to meningococcus, 1 due to

hemolytic streptococcus, and 1 due to Pneumococcus type XIX. All were proven by autopsy. One case of meningococcus meningitis had a twenty-eight hour history. Three cases of asphyxia were reported, one due to aspiration of gastric contents as shown by autopsy and two cases of strangulation (bed clothes), one proven by post mortem examination. There was only one sudden death due to prematurity who died after eight hours and whose autopsy showed prematurity and bilateral adrenal hemorrhage. The patient weighed one pound, 9 ounces.

The other causes were a case of methyl salicylate poisoning (twenty-two hours), a foreign body in the trachea with laryngo tracheitis (thirty hours), a staphylococcus albus septicemia (thirteen hours), a diarrhea and dehydration (twenty-four hours), a bulbar polio (twenty-four hours), and an erythroblastosis foetalis (twenty-three hours), all proven by post mortem examination.

There was one case of unknown cause of death (a nine days old white female) who had had a nose bleed 10 hours before and was brought into hospital D. O. A., no post mortem examination was done. There is no further information on the chart.

Cause	*No.*	*% SD*	*Aut.*	*Pr.*
Meningitis	6	20.0	6	6
Pneumonia	3	10.0	3	3
Asphyxia	3	10.0	2	2
Premature	1	3.3	1	1
Other	6	20.0	6	6
Unknown	1	3.3	0	0
Operative	10	33.3	10	10

In 1946 there were 222 deaths and 33 sudden deaths.

There were 5 deaths due to prematurity, 4 of whom had post mortem examinations; all of these showed atelectasis and 2 showed multiple hemorrhages. All weighed less than 4 pounds.

Four cases of congenital heart disease, all proven by autopsy, were reported. Two of these were cases of cardiac hypertrophy with dilatation and congestive heart failure (one a white female, six months old) (one a colored female, two months).

Four cases of pneumonia were reported, all proven by autopsy. Two dead on arrival, one living 20 minutes and one, 8 hours. The two who were dead on arrival and the case living, were three weeks, three months, and five days old respectively.

There were three cases of severe burns in this series.

Two cases of laryngo tracheo bronchitis—one had a post mortem examination and the other an ENT consultation and tracheotomy. The duration of hospitalization was four hours and one hour respectively.

There were two traumatic sudden deaths both due to skull fractures and cerebral hemorrhage and both proven by autopsy.

The other causes of death were diarrhea and dehydration (three months old colored female), Waterhouse Friederichson Syndrome (three and one-half white male), hemorrhagic disease of the newborn (six hours old white female). Erythroblastosis foetalis (twelve hours old white male), asphyxia due to plugging of bronchi with thick mucous (three years old colored female) and one case of acute glomerulo nephritis with congestive heart failure (ten months old colored male).

There was one case which we classified as unknown. A one year old colored female who was hospitalized for two hours. She had been sick for seven hours with a cough and several convulsions between 8:00 and 11:45 a.m. On post mortem examination she exhibited dilatation of the stomach, generalized lymphadenopathy, cerebral and meningeal edema.

Cause	*No.*	*% SD*	*Aut.*	*Pr.*
Premature	5	15.0	4	5
Cong. Ht. D.	4	12.0	4	4
Pneumonia	4	12.0	4	4
Burns	3	9.0	3	3
L. Tr. Br.	2	6.0	1	2
Trauma	2	6.0	2	2
Other	6	18.0	6	6
Unknown	1	3.0	1	?
Operative	6	18.0	6	6

In 1947 there were 195 deaths and 37 sudden deaths.

Prematurity accounted for 7 sudden deaths in 1947; 5 of these had autopsies; 3 showed atelectasis. The average hospitalization was six hours, the shortest being five minutes, and the longest twelve hours. All were under two and one-half pounds except for one who weighed four pounds nine ounces.

Congenital heart disease accounted for five of the deaths all proven by post mortem examination. One of these is a case of idiopathic cardiac hypertrophy with dilatation and congestive heart failure (11 days old colored male). Three of these cases died in congestive heart failure.

Three cases of Waterhouse Friederichson Syndrome were reported and all confirmed at post mortem examination. There were three cases of asphyxia, all of whom were dead on arrival. Two had autopsies showing suffocation.

There were two cases of laryngo tracheo bronchitis, both proven by autopsy, and two traumatic deaths, one an intraventricular hemorrhage and the other a rupture of the liver, kidney, and lung both proven at autopsy.

The other cases were, one case of extensive burns and smoke inhalation (one year white male), one of bilateral lobar pneumonia (colored female, fifteen months old), a pneumococcic meningitis who was dead on arrival (four months old white male) a meningitis of undetermined etiology (six years old white male), a phosphorus poisoning with congestive heart failure (two years old colored male) two cases of intracranial hemorrhage in new born (sixteen hours old colored male and a two hours old colored male), and an erythroblastosis foetalis (three days old white male). These were all proven by autopsy, and a case of massive atelectasis in a newborn proven by x-ray.

Cause	*No.*	*% SD*	*Aut.*	*Pr.*
Premature	7	19.3	5	7
Cong. Ht. D.	5	13.8	5	5
Wat. Fr. Syn.	3	8.3	3	3
Asphyxia	3	8.3	2	2
L. Tr. Br.	2	5.5	2	2
Trauma	2	5.5	2	2
Other	9	25.0	8	9
Operative	6	16.6	6	6

In 1948 there were 127 deaths and 26 sudden deaths. Prematurity accounted for three deaths, all proven by autopsy. Two showed atelectasis and one hemorrhagic disease of the newborn. All weighed less than three pounds eight ounces. There were four cases of laryngo tracheo bronchitis, all proven by autopsy. One was due to laryngo-tracheal diphtheria.

Three cases of trauma were reported, two were due to skull fracture and lacerations of the brain, one proven at post, and the other at operation, and one case of ruptured liver, kidney, and spleen proven by post mortem examination.

There were two cases of septicemia, one due to non-hemolytic staphylococcus albus and the other to a pseudomonas aeroginosa, both proven by autopsy and culture.

Cause	*No.*	*% SD*	*Aut.*	*Pr.*
L. Tr. Br.	4	15.2	4	4
Premature	3	11.5	3	3
Trauma	3	11.5	2	3
Septicemia	2	7.5	2	2
Wat. Fr. Syn.	1	3.8	1	1
Burns	1	3.8	0	1
Other	4	15.2	4	4
Unknown	1	3.8	0	0
Operative	7	27.0	7	7

There was one Waterhouse Friederichson Syndrome proven by post mortem examination and one case of extensive burns, 2° and 3° of 55 per

cent of the body surface. The other cases are two of diarrhea and dehydration, one of hemorrhagic disease of the newborn and one of a non-hemolytic streptococcus hepatitis in a newborn all proven at autopsy.

There is one case for which no cause of death is known. That of a one and one-half month old colored female who was dead on arrival with the only history being a nose bleed one hour prior to admission. No autopsy was done. During attempts at artificial respiration, a yellow material resembling vomitus was noted to flow from the nose.

We have also included in this analysis cases who died either during surgery or in the twenty-four hour post-operative period. These we have indicated in the table grouped according to the type procedure and the time of death in relation to surgery.

The first group is neurosurgical. There were fifteen deaths in this group. Of the eleven who died during surgery, five were pneumo encephalograms, one showing an air embolus of the right heart and pulmonary artery and two showing atelectasis. All had autopsies. The other six cases were three subdural hematomectomies, two craniotomies for hydrocephalus, and craniosectomy for microcephalus. All had autopsies, two showing atelectasis. Except as mentioned the autopsies revealed only the pathologic process mentioned and the surgical procedure.

There were four deaths in the three hour post-operative period. One a subdural hematomectomy dying three hours post-operatively in whom the post showed atelectasis. A craniosectomy for microcephalus who died two hours post-operatively. A craniotomy for hydrocephalus who died fifteen minutes post-operatively in whom the autopsy showed multiple adenomata of both adrenal cortices, and a pneumoencephalogram dying thirty minutes post-operatively.

Under the abdominal group we have fourteen deaths. Of these seven died in the operating room. An appendectomy, who on post mortem examination, showed acute rheumatic fever and pneumonia. A perineal section for imperforate anus in which case the post mortem examination showed an absence of the right kidney and a left hydronephrosis, a strangulated hernia showing acute right heart failure, atelectasis and gastric dilatation, a Wilms' tumor and metastasis to the lungs and liver and an intraventricular septal defect. A hernia showing atelectasis, and a strangulated hernia with aspiration of gastric contents.

Two cases died in the three hour post-operative period. One a strangulated hernia dying three hours post-operatively showing atelectasis, and the other, a volvulus with intestinal obstruction dying two hours post-operatively showing atresia of the ileum.

There were five deaths in the twenty-four hour period. Two were due to

intussusception, one showing atelectasis and gangrene, the other a mesenteric artery thrombosis dying twenty-eight hours post-operatively respectively. One of ruptured appendix with peritonitis, dying six hours post-operatively.

A pyloric stenosis, dying in eight hours showing atelectasis, and a volvulus with intestinal obstruction, who died four hours post-operatively.

In the ear, nose, and throat classification we have five cases, four dying in the operating room.

One dying during anaesthesia prior to a tonsillectomy and adenoidectomy who had no autopsy, another during anaesthesia prior to bronchoscopy in whom the post showed pneumonia, bronchiectasis, nephrosis, and atelectasis. A laryngeal diphtheria dying immediately after bronchoscopy in whom autopsy showed respiratory obstruction by a membrane and right heart failure, and an acute laryngo tracheo bronchitis in extremis even with a tracheotomy who died during bronchoscopy instituted in an attempt to save his life when the patient developed marked respiratory difficulty.

One case of acute laryngo tracheo bronchitis died two hours after tracheotomy. The autopsy showed atelectasis.

Four patients undergoing thoracic surgery died during operation.

One a tetralogy of Fallot the autopsy showing cerebral thrombosis and infarction.

An incarcerated diaphragmatic hernia and intestinal obstruction who had no autopsy.

A closure of a patent ductus arteriosus who on autopsy showed chronic myocarditis and hemorrhage from the pulmonary artery.

And an esophageal atresia and tracheo esophageal fistula showing aspiration of gastric contents, pneumonia, and atelectasis on autopsy.

The plastic case is that of a cavernous hemangioma of the neck that died on the table in shock.

Over this six year period there were 1,345 deaths, 200 of which fit our criterion of sudden death.

Operative deaths—hours post-op.

Type	*OR*	*3 Hrs.*	*24 Hrs.*	*Total*
Neuro	11	4	0	15
Abdo	7	2	5	14
EENT	4	1	0	5
Thoracic	4	0	0	4
Plastic	1	0	0	1
Total	27	7	5	39

This represents 14.9 per cent of all deaths. On this table we have included the autopsies for each year and percentage autopsied as a matter of interest.

Of the 200 sudden deaths, we consider 188 or 94 per cent proven, 175 by autopsy, the other 13 by other means.

	Totals				
Year	*TD*	*SD*	*%*	*Aut.*	*%*
1943	279	38	13.6	216	77.6
1944	266	37	13.9	204	76.6
1945	256	30	11.6	208	80.6
1946	222	33	14.9	177	79.7
1947	195	36	18.4	156	80 0
1948	127	26	20.5	109	85.8
Total	1345	200	14.9	1,070	78.5

There were 31 of the sudden deaths who were dead on arrival.

For the six year period 39 or 19.5 per cent of the sudden deaths fit our criterion of operative deaths.

	Totals				
Year	*SD*	*Aut.*	*Pr.*	*DOA*	*OP*
1943	37	31	33	3	6
1944	37	28	35	4	5
1945	30	28	28	4	10
1946	33	31	31	6	6
1947	37	34	36	7	6
1948	26	23	25	7	7
	200	175	188	31	39
%		87.5	94.0	15.5	19.5

Fifteen per cent of the sudden deaths were due to prematurity, 7 per cent to Waterhouse Freiderichson Syndrome, 6.5 per cent to pneumonia, 6.5 per cent to congenital heart disease, laryngo tracheo bronchitis, 6 per cent, asphyxia, 5.5 per cent, meningitis and trauma, each 5 per cent and burns, 4 per cent.

Cause	*No.*	*% of 200*
Operative	39	19.5
Premature	30	15.0
Wat. Fr. Syn.	14	7.0
Cong. Ht. D.	13	6.5
Pneumonia	13	6.5
L. Tr. Br.	12	6.0
Asphyxia	11	5.5
Meningitis	10	5.0
Trauma	10	5.0
Burns	8	4.0
Diarrhea	7	3.5
Septicemia	6	3.0

DENTIGEROUS CYST

Case Report No. 174

Bennett Olshaker, M.D. R. T. 42–9854

R. T., a six year old Negro boy, was admitted to The Children's Hospital on January 25, 1949. Six weeks prior to this he had fallen, striking his face against the ground. A school physician, during a routine examination of the boy two days after the fall, noted a small mass in the left maxillary region and advised that the patient be brought to the hospital for study.

The child was first seen in surgery clinic three weeks after the school examination. A "moderately fluctuant, non-tender mass in the left maxillary region" was described and the impression was that it represented a liquefying hematoma. One week later he was seen in the otorhinolaryngology clinic and the impression was that the mass represented a cyst which was either "neoplastic or dental in origin." The report of a roentgenological examination made two weeks before admission to the hospital stated that "in the lateral view there is a well circumscribed shadow with the periphery calcified which is situated just below the nose in the region of the upper lip. The exact nature of this is not clear; it is not suggestive of a malignant lesion."

Physical examination on admission revealed a boy who did not appear ill. His rectal temperature was 99.2 F., pulse was 80 per minute, respirations 20 per minute, and blood pressure was 105/65. Small freely movable posterior cervical, axillary, and inguinal lymph nodes were felt. There was a hard, non-tender, immovable mass palpable in the maxillary region. It measured approximately 2.5 by 4.0 cm., seemed to be part of the bone and not attached to the submucosal tissue, and was located mainly to the left of the midline, extending, however, across the midline for about 1.5 cm. The only other positive physical findings were a sinus arrythmia and a soft systolic murmur which was heard at the left border of the sternum in the fourth interspace.

Hemogram showed a hemoglobin of 12.5 grams, 4,700,000 erythrocytes, and 4,150 leukocytes, 65 per cent of which were polymorphonuclear cells, 34 per cent lymphocytes, and 1 per cent eosinophiles. No abnormality was noted in the urinalysis. Kahn and Mazzini tests were negative. The serum calcium was 10 mgm. per cent and phosphorus 5.7 mgm. per cent. Alkaline phosphatase was 11.2 King-Armstrong units (Normal 6–20) and acid phosphatase was 4.3 King-Armstrong units (Normal 1–4). Bleeding time was one minute and coagulation time was three minutes.

Repeat roentgen examination of the maxillary region revealed "a zone of condensation around the previously described cystic-like lesion, which may indicate an encysted periosteal hemorrhage" (see figure 1). Examination of the chest and of the long bones of the upper and lower extremities demonstrated no evidence of abnormality.

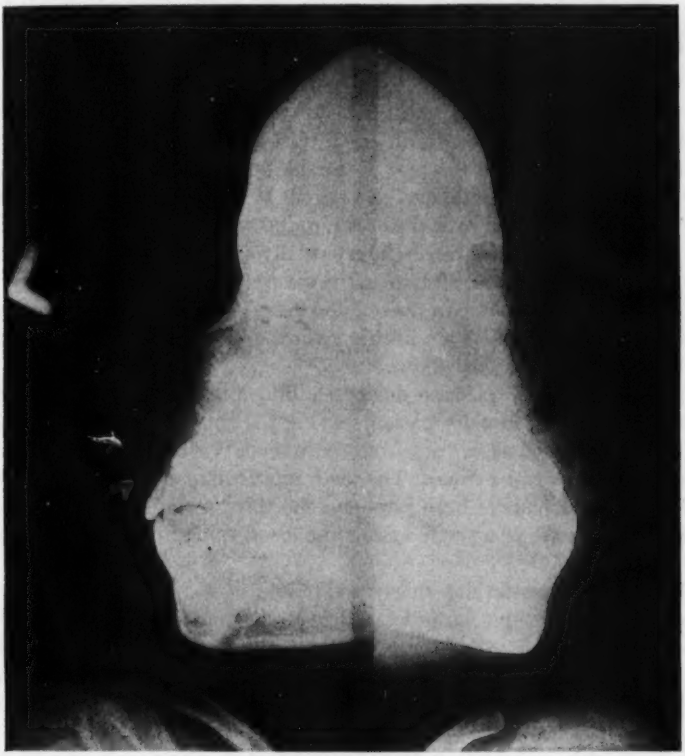

FIG. 1 R. T. Cystic like lesion of the maxillary bone surrounded by zone of calcification.

The impression of various examiners included hematoma, dentigerous cyst, and osteoma.

On February 25, 1949, an operation was performed. The operative note is as follows: "A cyst-like structure measuring approximately 3 cm. in diameter was removed from the left maxilla. The pedicle was attached to the left upper canine tooth which was also removed with the cyst. There

was another encapsulated tooth posterior to and connected with the cyst; this was also removed with the cyst. A plain gauze packing was inserted into the cavity and three catgut sutures to partially close the cavity."

Post-operatively, the patient received 300,000 units of procaine penicillin in oil daily for five days. He was discharged to be followed in the clinic. One month after the operation the cavity had closed and the patient was doing well.

DISCUSSION

A dentigerous cyst is one which contains the crown of a partially or completely formed unerupted tooth in a thickened fluid-distended follicle. Many appear to arise at the root of the deciduous predecessor. The lesion may begin as a small dental cyst at the root of a deciduous tooth but later may enlarge and extend over the crown of the underlying permanent tooth and progress in an apical direction eventually enclosing a varying amount of the permanent tooth. Occasionally several unerupted teeth may be enveloped. The cyst is circumscribed and generally covered by a thin layer of bone within which there is a fibrous layer that may be lined with granulation tissue or cuboid epithelium.

Usually this tumor is found during childhood or adolescence, but cases have been reported in older patients where the third molar was involved. The ages of reported cases vary from six to seventy-three years. Sometimes a familial factor can be traced. The teeth most commonly involved are the canines and premolars of both jaws and the third molars of the mandible. While the presence of a single cyst is the usual thing, multiple cysts have been observed.

Most of the cysts develop slowly and painlessly as a superficial tumor in the outer side of the alveolar process. Rarely they may extend for a considerable distance reaching the floor of the orbit, the zygomatic arch, ascending ramus, and coronoid process. The bony capsule may be so thin that a celluloid crackling is produced when pressure is put upon it. Fluctuation may sometimes be detected.

Radiographic examination discloses a definitely circumscribed cyst, the contents of which are radiolucent and the periphery well defined and radiopaque. Contained within the cyst is the crown of a partially or completely formed tooth.

When the cyst is small the treatment may consist of complete excision. Larger ones may be treated by removal of the outer wall and curettage of the remaining cavity followed by cauterization with phenol and neutralization with 95 per cent alcohol following which the cavity can be packed with gauze. Adjacent teeth sometimes become devitalized and require extrac-

tion. However, if the operation is performed in which the buccal wall of the cyst together with the alveolar bone is removed without disturbing the unerupted teeth, quite often the teeth will erupt and be functional.

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CONGENITAL URETHRAL STRICTURE

Case Report No. 176

William M. Crowell, M.D.
Robert H. Anderson, M.D.

Congenital urethral stricture is a frequently overlooked cause of urinary tract obstruction in children. Emphasis is placed on early diagnosis and treatment as a means of preserving kidney function and even life itself.

Case Report G. C. T. 49–10988

This seven week old white male was admitted on September 5, 1949 with a history since birth of intermittent diarrhea which was treated with sulfonamides twice a week for periods of two days each. Urination had always been in small amounts. He had hematuria once and vomiting two or three times eight days before admission. He became drowsy the week before hospital entry and abdominal distension was present five days before admission. He was hyperirritable the day before and had a generalized convulsion on the morning of admission.

Past history and family history were non-contributory.

Physical examination on admission revealed a stuporous, afebrile, and moderately cyanotic infant. There was abdominal distension with ascites. A firm mass which was 4 by 5 cm. in size and which seemed separate from the liver could be felt in the right upper quadrant of the abdomen. A partial stricture of the rectum was also found.

The patient was immediately put in an oxygen tent. An intravenous pyelogram showed ascites and no filling of the pelves or calyces. A non-protein nitrogen determination on September 7 was 132 mgm. per cent. An abdominal paracentesis was done on September 8 with the removal of 600 cc. of clear, straw-colored fluid. The patient developed distension of the bladder and had to be catheterized repeatedly. A cystogram was done on September 10 (figs. 1 and 2). On September 12, his temperature rose to 104.0 F. and an E. coli urinary tract infection was found which was effectively treated with sulfonamides and chloromycetin. The non-protein nitrogen was 94 mgm. per cent on September 12. At this time a catheter could not be passed so a suprapubic cystotomy was performed. The patient voided only a few cubic centimeters post-operatively and expired on September 22. An autopsy was performed.

In brief, the necropsy findings were pulmonary edema, fatty infiltration of the liver, congestion of the spleen, minimal cystic fibrosis of the pancreas, and thrombosis of the meningeal and superficial cortical veins. The urinary findings were bilateral hydronephrosis, pyonephrosis, pyelonephritis, hydroureters, and hypertrophy of the bladder, all of which were secondary to an

annular stricture of the prostatic urethra, the diameter of which was less than 1 mm. Histologically, the cystic areas in the kidneys (fig. 3) were

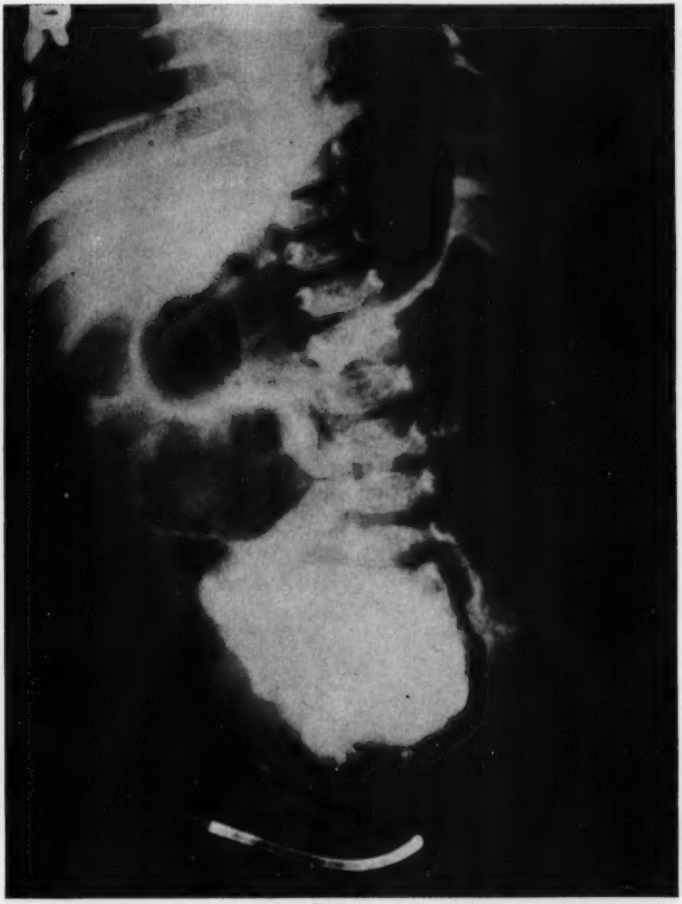

FIG. 1

FIGS. 1 AND 2 G. T. Note marked hydro-ureters and enlargement of the bladder.

composed of necrotic tissue and compressed, sclerotic tubules. The glomeruli were hypoplastic and infection with abscess formation was present.

DISCUSSION

If the urinary obstruction had been discovered earlier, this patient could have been treated successfully, but he was admitted in uremia with ap-

parently little kidney function remaining. In this case, the necropsy findings were those of chronic urinary tract obstruction, i.e., hydronephrosis with secondary infection. The histological picture of dilated tubules and

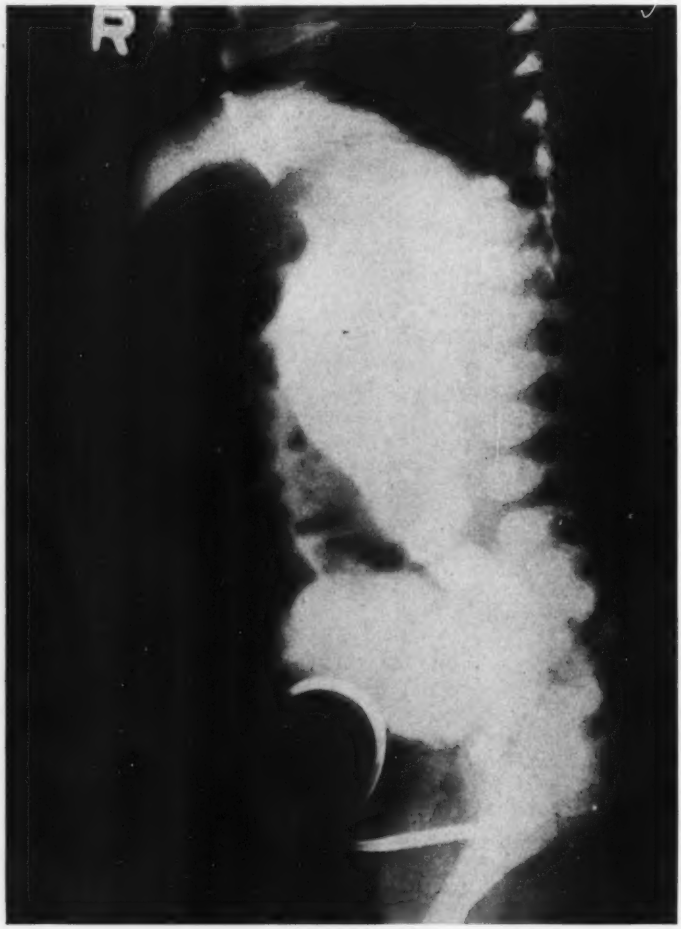

FIG. 2

hypoplastic glomeruli is typical of acquired hydonephrosis secondary to urethral obstruction.

Hydronephrosis is the pathologic end result of any chronic urinary tract

obstruction. An obstruction of the urethra results in bilateral kidney involvement, the prognosis of which, if untreated, is grave. Unilateral hydronephrosis may be secondary to ureteral obstruction. Of one hundred cases of hydronephrosis in children reported by Kretschmer[1], sixteen were due to obstruction of the urethra or the bladder neck and all of these were in males.

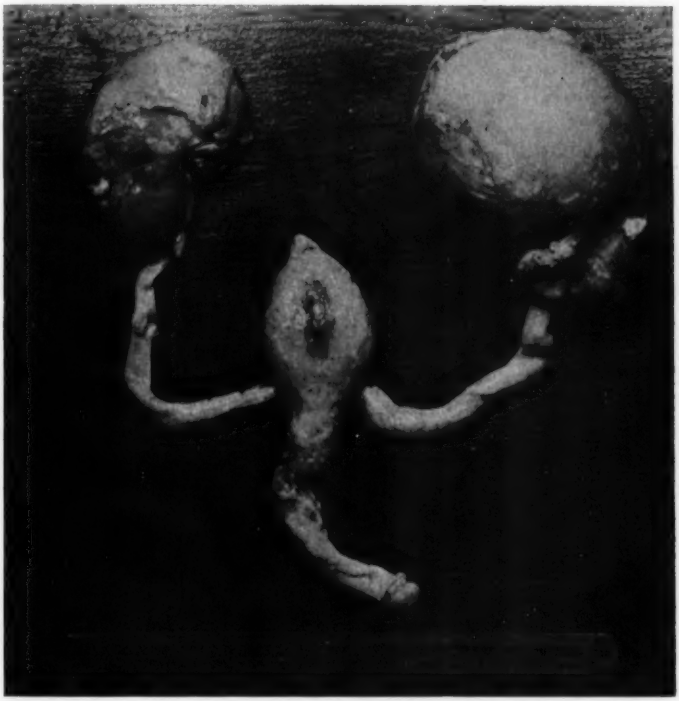

FIG. 3 G. T. Autopsy specimen of the entire urinary tract showing the particularly large cyst of the left kidney which is secondary to the urethral stricture.

Classification: Lesions obstructing the urethra or the vesical neck in children are generally classified as follows:

I. Neuromuscular Dysfunction
   A. Spina bifida
   B. Spinal cord disease
   C. Peripheral nerve involvement

II. Mechanical Obstruction
  A. Intrinsic Lesions
    1. Strictures
      a. Annular
      b. Valves
      c. Bands
    2. Lesions of the Internal Sphincter
      a. Sclerosis and contracture
      b. Hypertrophy
      c. Spasm
    3. Lesions of Verumontanum
      a. Hypertrophy[2]
      b. Congenital cysts[3]
  B. Extrinsic Lesions
    1. Prostatic Lesions
      a. Adenoma[4]
      b. Hypertrophy with pedunculation
      c. Sarcoma
      d. Myoma[5]
    2. Periurethral Sarcoma[6]

Congenital strictures of the urethra can be divided into three types. The first is annular stricture with involvement of all layers of the canal. Secondly, there are valvular strictures which are usually diaphragmatic in type; they may be multiple and are most common in the region of the verumontanum. The third type is bands which are usually elevations of the mucosa and are most common in the membraneous urethra. Strictures may be multiple since the urethra embryologically is derived from three anlage, the junctions of which may be possible sites. Valves may be present with their concavity toward the bladder. These are not encountered on sounding but still may be obstructing urination.

The posterior urethral valve is by far the most common congenital stricture. As of 1940, one hundred and five cases had been reported.[7] The first clinical recognition of congenital urethral valves was by Hugh Young in 1912, and the first exhaustive review of the literature followed with Young as one of the co-authors.[8]

Diagnosis: The signs and symptoms of obstruction in this portion of the urinary tract can be divided into three categories: those due to the actual obstruction, those due to uremia, and those due to residual urine. Those due to the obstruction include difficulty in starting micturition, dribbling, and incontinence. Uremia may produce malaise, headache, malnutrition, recurrent respiratory infection, diarrhea, vomiting, convulsions, or coma. Residual urine causes a distended bladder, hydro-ureters, and hydronephrosis; it is usually accompanied by infection which is demonstrated by pyuria, fever, back pain, abdominal pain, and dysuria.

Once signs of obstruction are discovered, the next recommended step is intravenous urography which in turn should be followed by cystoscopy and cystography. Retrograde urography is contraindicated because it increases the incidence of urinary infection. Definitive treatment should be carried out by a competent urologist, but it remains the duty of the pediatrician to discover these cases and to make a tentative diagnosis. Congenital strictures are frequently overlooked and their frequency of diagnosis can be increased by the thorough investigation of persistent urinary symptoms.

There is one group [9] of rather uncommon cases due to neuromuscular dysfunction which occurs only in male children. The pathology is that seen in other cases of obstruction, but no obvious obstruction is found. The spinal cord of the sacral region, however, shows an infiltration of inflammatory character near the anterior horn cells and in some cases, delayed myelinization.

In making a diagnosis, it is well to remember that there are frequently other associated congenital anomalies[3], that the conditions causing urethral obstruction are practically always found in male children, and that these children usually present no evident urinary abnormality at birth[10].

SUMMARY

1. A fatal case of annular urethral stricture in a seven week old white male is presented.
2. Classification of congenital urethral obstruction in children is given with a brief discussion of the types and their recognition.
3. Coincidental congenital anomalies frequently occur.

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ATROPINE POISONING

Case Report No. 176

William L. Sager, M.D.

P. B., a two year old colored female was admitted to Children's Hospital on November 5, 1949 with the complaint of unusual behavior for two hours prior to admission. The child had been perfectly well until the morning of the day of admission. At this time she fell off her mother's bed. She cried immediately afterwards and seemed to be all right when her mother picked her up. There was no period of unconsciousness and no vomiting. The patient returned to her play after this incident. A normal lunch was eaten and she appeared well during play in the afternoon. She lay down for a nap at 5:00 P.M. and awoke at 8:00 P.M., crying. The patient's mother tried to get her to walk, but the child staggered, leaned to the right, and needed support to stand. She was put to bed again, and she dozed off. Within five minutes she cried out, throwing up her arms and legs, as if startled. At this time, she was also noticed to make purposeless movements with her hands—to grab at non-existing matter, to put it into her mouth, and to chew on same. The child began to ask her mother to make her older sister stop hitting her. In reality, her sister was not near the patient. Because of the peculiar behavior of the two year old, she was brought to the Dispensary and subsequently admitted to the hospital at 9:45 P.M. The patient lived in the city. There was no history of the ingestion of any material.

The past history and family history were non-contributory to the present illness.

Physical exmination at the time of admission revealed a well developed, well nourished colored female of two years, who appeared acutely ill. Her temperature was 99.0 F.; the pulse rate was 64, and the respiratory rate was 24. Blood pressure was 100/70. She presented bizarre mannerisms. Sitting in bed, she moved her arms about slowly and aimlessly. She would grasp at imaginary objects and put them into her mouth. Throughout the examination, she would scream, then throw forward her arms and legs, as if to keep herself from falling backward. The child spoke in single words, saying, "hello," "goodbye." She repeatedly pointed to the window and exclaimed, "doggie." The pupils were large, fixed, and equal. They did not respond to light or accomodation. Eye grounds were examined without difficulty and the disks were normal. The patient did not blink her eyes nor did she attempt to turn her head away from the light. No nystagmus was present. Neurological examination was normal so far as could be deter-

mined. The child fell toward her right side on trying to walk. Her cheeks were flushed. Physical examination was otherwise negative.

The laboratory examination revealed a hemoglobin of 14 grams, with 4.7 million red blood cells. The white cell count was 8,000 with 57 per cent neutrophiles and 39 per cent lymphocytes. The urinalysis, serology test for syphilis, O. T., Schick tests, and sickle cell preparation were negative. Spinal tap revealed two white cells with clear fluid and normal pressure.

Several hours after admission, the child's mother telephoned to inform us that she had found empty a bottle of eye-drops that had been prescribed for her child at Eye Clinic that same day. The prescription was located and found to be one dram of 1 per cent atropine sulfate. Upon receiving this information the child's stomach was lavaged with warm water. She was ordered phenobarbital gr. $\frac{1}{4}$ (H) stat and every four hours for restlessness, and pilocarpine, 5 mg. stat and every one hour for two doses. Fluids were forced and the child took them eagerly.

Throughout the night she continued to behave in the unusual manner previously described. She would doze for about five minutes, then scream and appear startled. Her temperature, pulse, and respirations were checked frequently during the period of hospitalization and they varied little. The pulse rate went as high as 120. The temperature fluctuated between 99.0 and 100.4 F. Respirations were never lower than 20, and were 32 at their peak. Because the child's mouth and throat did not seem especially dry and because she took fluids well, pilocarpine was discontinued after two doses.

By daylight, the patient's odd behavior began to subside. She fell asleep until noon, at which time she awoke, drowsy but calm. Toward afternoon, she began to talk coherently, stood up in bed, and looked around. Pupils at this time were less dilated than on admission and reacted sluggishly to light and accomodation. Her further recovery was uneventful and she was discharged from the hospital in an improved condition.

DISCUSSION

This case was of interest mainly because of the bizarre picture that the child presented on admission to the hospital. Many possibilities were mentioned by various members of the House Staff in an attempt to explain the patient's condition. Brain tumor, cerebral hemorrhage, an encephalitis, a blood dyscrasia, and alcoholism were some of the diagnoses entertained. The fact that a negative history of the ingestion of any material was obtained seems pertinent, for ingestion may be the case, regardless of the extent of the informant's knowledge. The index of suspicion should always be high for the ingestion of any material by children under the age of three years.

Acute atropine poisoning usually results from excessive doses of atropine or drugs containing atropine. Dangerous symptoms have resulted from grain one-twentieth to grain one-tenth, and death has occurred in six hours after it was taken. Nine out of the ten cases of atropine poisoning on record at The Children's Hospital were the result of the accidental ingestion by children. There were no deaths, however. Morton[1] reported that only two of the eight cases seen at Duke Hospital from 1930 to 1938 were due to ingestion by children. Two fatalities occurred there, both following the use of 1 per cent atropine sulfate solution as eye drops and eye ointment.

The earliest and most characteristic symptoms of atropine poisoning are:

1. Excessive thirst, dryness of mouth and throat.
2. Flushed skin, dry, especially on face and neck.
3. Widely dilated pupils.
4. Very rapid pulse.
5. Rapid respirations, later slow and shallow.

If very large doses of atropine are taken, these symptoms are increased and may be followed by loquacity, delirium, jerky movements—the so-called "belladonna jag." Atropine in large doses is a respiratory stimulant. Poisonous doses may depress the respiratory center, finally paralyzing it, with death ensuing. Fever occurs in approximately 10 per cent of the cases, according to White[2]. One out of the ten cases at The Children's Hospital had a high fever (104.0 F.). Two other cases at this hospital had a temperature of 101.0 F.

The following table shows the usual progression effect of therapeutic and toxic doses (After Schmiedeberg[3]):

Milligram	*Grain*	*Symptoms*
0.5	1/120	Slowing of pulse and very slight dryness of throat
0.5–1.0	1/120–1/60	Dryness in mouth, often with thirst
2.0	1/30	Pupil dilated, not quite immobile
3–5.0	1/20–1/12	Headache. Dysphagia. Alteration of voice. Muscular weakness. Restlessness
7.0	1/10	Considerable dilatation of pupils. Disturbance of vision
8.0	1/8	Excitement and muscular incoordination more marked
10.0	1/6	Apathy. Hallucinations or delirium. Unconsciousness

The usual fatal dose has been stated to be 0.1 Gm. or more for adults and 0.01 Gm. for children. Assuming that our patient ingested all of the one dram bottle of 1 per cent atropine sulfate solution, she would have taken 30 mg. (One drop of the usual 1 per cent atropine sulfate solutions contains 9.5 mg.) Pilcher[4] demonstrated a wide variation of tolerance to

atropine in children. One child tolerated 16 mg. a day while another reacted to one drop (0.05 mg.) of this same solution.

The treatment of atropine poisoning is primarily three-fold.

1. Stomach lavage as soon as possible. Five per cent tannic acid or a one per cent potassium iodide solution is recommended[5]. If lavage is impractical, suitable emetics should be used.

2. Parasympathetic drugs should be given. Pilocarpine is the logical physiological antidote because it acts antagonistically to atropine on the autonomic nervous system. Half-hour doses of 5 to 10 mg. of pilocarpine may be given until the mouth and throat are moist.

3. Sedation should be used carefully. It is indicated only in the early stages of atropine poisoning and should be discontinued once depression has set in.

High fevers should be relieved by the usual methods. Strychnine and caffeine may best meet the respiratory and circulatory failure. If the patient passes small amounts of urine, catheterization should be done frequently to avoid reabsorption of atropine from the urine in the bladder.

Morphine is contra-indicated in the treatment of this type of poisoning. Although atropine is the antidote for morphine, the dangerous effect of atropine is due to the exhaustion of breathing. If morphine is given in such cases, the respirations are only made slower.

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CLINICO-PATHOLOGICAL CONFERENCE

Directed by: E. Clarence Rice, M.D.
Assisted by: William M. Crowell, M.D.
Francis J. Troendle, M.D.
By Invitation: John A. Washington, M.D.

William M. Crowell, M.D. W. H. 39-7040

W. H., a white male was born on May 26 by normal hospital delivery of a term delivery of a term pregnancy. The birth weight was eight pounds, thirteen ounces, and the child was breast fed. The vitamin intake was considered adequate. The family history was non-contributory and the parents' serology was reported as being negative. There was one older sibling of three and one-half years who was living and well and one deceased sibling who died at five weeks of age from bronchopneumonia.

At the age of eight weeks, prior to which time the child had been perfectly well, a hospitalization of two weeks was required at another hospital for "bronchitis."

His first admission to this hospital was on September 12 at the age of three months, with the chief complaint of vomiting of ten days' duration. The vomiting was described as being intermittent (two to seven times daily), at times green, post-prandial zero to three hours, and at first projectile but not so at the time of admission. During this time he was having two to three soft stools daily. Phenobarbital had been prescribed unsuccessfully elsewhere. He was breast fed until this admission. The mother thought he had lost about one and one-half pounds, the admission weight being eleven pounds, ten ounces. On initial examination, no infection was present to explain the fever of 100.8 F.; no abdominal masses were palpated, and the only positive findings were frontal bossing and thickened epiphyses the interpretation of which was rickets.

Minimal vomiting occurred during the first week and he gained weight, then severe non-projectile vomiting began and x-rays suggested a delayed gastric emptying time with diminished peristalsis. One examiner thought a small mass was palpable in the right lower quadrant, but it is was not substantiated. During the next two weeks the vomiting persisted but was much less frequent. Thick feedings with atropine were required together with parenteral fluids but in spite of this, the child became poorly nourished and edematous (possibly due to excessive parenteral saline). By the fourth hospital week a low grade fever was present (99.0 F. to 101.0 F.) and a bilateral myringotomy was required for purulent bilateral otitis media due to C. hoffmannii. Concomitantly a diarrhea began with ten to fifteen yellow

dark green liquid stools daily. Repeated cultures revealed no organisms of the typhoid-dysentery group.

Coughing began by the fifth hospital week and was accompanied by course rales and bronchi in the chest but these and the low grade fever subsided spontaneously in three or four days. A chest x-ray showed "increased markings suggestive of upper respiratory infection or sinus disease."

By the seventh week the chest findings persisted as well as the poor general condition, so the patient was given a four day course of sulfapyridine after which the cough and diarrhea ceased and feedings were retained. The patient was discharged during the ninth hospital week on November 8. The diet consisted of a simple evaporated milk formula, and supplemental vitamin D. The discharge weight was ten pounds, fifteen ounces. During the admission several tuberculin tests were negative, urinalyses were compatable with the low grade urinary tract infection which was thought to have occurred during the fourth week; a Kahn test was negative and repeated hemograms showed a leukocytosis averaging 10,000 cells per cubic millimeter, with neutrophilic granulocytes ranging from 48 to 78 per cent of which about 75 per cent were segmented. The erythrocytes increased from 3,310,000 per cubic millimeter with 10.5 grams of hemoglobin on admission to 5,000,000 with 14 grams of hemoglobin on discharge.

The second admission (November 12 to 21) was four days after discharge from the first hospital admission. During the interval at home, fever, rhinitis, cough, ear tugging, and vomiting developed. The weight was ten pounds, eight ounces, the temperature was 102.4 F., the ear drums bulged, the chest was normal, and the skin was dry. Fluids and sulfanilamide were used effectively and the patient was discharged as well, weighing ten pounds, seven ounces. A transfusion was required for anemia.

The third admission to this hospital was two weeks later at six months of age from December 3 to 16 and the admitting complaint was diarrhea, vomiting, coughing, and grunting respirations for which steam inhalations had been used unsuccessfully the previous night. The physical examination discolosed an acutely ill, febrile, underdeveloped child who had an expiratory grunt and exhibited air hunger. The skin was sallow and was questionably cyanotic. Course ronchi were heard throughout the chest. Examination of the abdomen was negative. An acute tonsilitis and left otitis media were associated findings. Later that day the breath sounds were reported as being distant and there was definite sternal retraction. On December 5, laryngoscopy was negative in spite of the patient's poor condition. Bronchoscopic aspiration was followed by some relief but no etiological diagnosis was made. Slow but spontaneous recovery then took place. Cultures were positive for pneumococcus and negative for C. diphtheriae. A chest x-ray showed obstructive emphysema which indicated a high but un-

explained obstruction. Treatment consisted of sulfanilamide, oxygen, and general supportive measures. On discharge the weight was eleven pounds, four ounces and the condition was excellent.

The fourth admission was on January 9 at which time the patient was seven months old. The interval three weeks' history was negative. The admitting complaint was fever with respiratory difficulty of two days' duration and the physical examination disclosed a bluish mottling of the skin, a moderate tonsillitis, a bilateral otitis media, harsh breath sounds with scattered rales, substernal and intercostal retraction, and a slightly distended but otherwise negative abdomen. The temperature was elevated to 104.0 F. and went to 105.0 F. the following day although the child was somewhat improved. Specific therapy was sulfanilamide and the patient was discharged on January 19 with the diagnosis of acute laryngo-tracheobronchitis. The blood count on admission was 3,200,000 erythrocytes per cubic millimeter with 10.5 grams of hemoglobin and 7,700 leukocyte count with 62 per cent lymphocytes. The urine was negative.

The fifth and last admission was on March 9 at which time the patient was eight months of age. The complaints were fever of twenty four hours' duration, noisy respirations which had become worse, and cough. The temperature was 104.0 F. and the remaining findings were bilateral otitis externa, a bright red pharynx filled with tenacious mucous, tendency to pigeon breast deformity, dyspnea with a long difficult expiratory phase, a few rales, and abdominal distension. The temperature rose to 105.0 F. and rales became audible throughout the chest. The breath sounds became diminished in both bases with some dullness to percussion in the right base. Again the acute episode was controlled by sulfapyridine and oxygen only to have a recurrence of the fever a few days after the sulfapyridine was discontinued. Chest x-rays at this time revealed superior mediastinal widening and a thoracotomy was advised. The month before operation was spent in preparing the patient for surgery. At the time of operation on April 20, the temperature was normal; however, the child still required oxygen, still had dyspnea with bubbling rales and still had respiratory stridor. During anesthesia induction the child expired.

DISCUSSION

John A. Washington, M.D.: My impulse is to approach this case history backwards. That is, to assume that the superior mediastinal widening found in the final x-ray represented a local lesion which caused the train of respiratory tract symptoms leading to death. I have seen teratomas, neuroblastomas, and lymphosarcomas of the mediastinum. I cannot see how such a tumor could have caused so much trouble and have been invisible in previous x-rays or how it could have caused repeated episodes of

pneumonitis responding to chemotherapy without causing some stridor yet no stridor is mentioned except on the one admission when laryngo-tracheo-bronchitis was diagnosed. In this connection the x-ray reading of "obstructive emphysema due to a high but unexplained obstruction" must be considered. Certainly emphysema is characteristic of low obstruction either of the bronchioles, as in asthma and bronchitis, or of bronchial obstruction by a foreign body where a ball valve effect is operative. Bronchi dilate on inspiration when the lung is expanded by the pulling of the chest walls and air enters past an obstruction which on expiration blocks the passage. High obstructions such as laryngitis cause inspiratory stridor. Tracheal stenosis usually causes both inspiratory and expiratory stridor. Therefore, it would seem more reasonable to suppose that the generalized emphysema here was a result of bronchitis.

Tuberculosis was eliminated as a possibility by tuberculin tests.

Now let us approach this case from its beginning. Although it is stated that the family history is non-contributory, the death of a sibling at five weeks of age from bronchopneumonia suggests immediately that cystic fibrosis of the pancreas may run in this family. While five weeks is young for pulmonary complications to develop, it seems possible that a fatal bacterial pneumonia may have developed secondary to earlier than usual bronchial embarassment. It is now believed that the fundamental defect is the production throughout the body of inspissated mucous which in the smaller bronchi causes obstruction and leads to secondary infection. The patient's history states that there was bronchitis first at eight weeks of age. At three and one-half months, an episode of vomiting occurred and at four and one-half months there was diarrhea, otitis media, and coughing with rales in the chest. X-ray showed increased root markings. Sulfapyridine was effective in curing infection. Poor nutrition was evident. At five months, an upper respiratory infection occurred with vomiting. At six months this was repeated with diarrhea and the x-ray showed emphysema. This time sulfanilamide stopped the infection. At seven months laryngo-tracheo-bronchitis was diagnosed with recovery. Then came the final admission at eight months and death at nine months. There was dyspnea with a prolonged expiratory phase, tendency to pigeon breast deformity, rales, and signs of pneumonia.

This is a fairly typical story of cystic disease of the pancreas. If this diagnosis is correct, the case would fall into Anderson's group II (respiratory symptoms before six months) and in the subgroup which, instead of showing foul stools, shows repeated attacks of diarrhea. The thing which I cannot find out is whether a baby in this group can live for nine months and still not have foul stools. Nelson states that foul stools occur usually by the sixth month. In our case, no mention is made of stools in the latter part of

the child's life. It should be pointed out that the use of sulfapyridine dates this case. In the late 1930's, knowledge of cystic disease of the pancreas was meager.

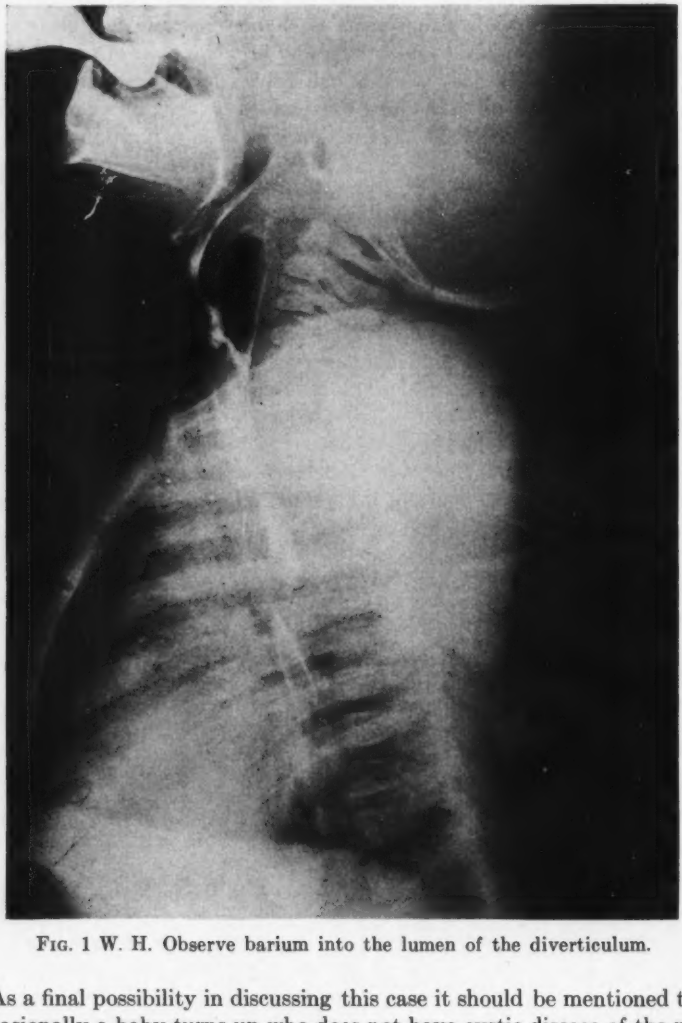

FIG. 1 W. H. Observe barium into the lumen of the diverticulum.

As a final possibility in discussing this case it should be mentioned that occasionally a baby turns up who does not have cystic disease of the pancreas, but who does have repeated attacks of bronchitis and succumbs to

bronchiectasis with abscess formation. Nine months is early for this sort of process to complete itself, however.

In summary I would place cystic fibrosis of the pancreas as by far the most likely diagnosis. The other conditions mentioned are possibilities.

PATHOLOGICAL DISCUSSION

E. Clarence Rice, M.D.: I believe that many of us had the same diagnostic possibilities come to mind as did Dr. Washington. My first impression after reading over the summary of the case record was to come to the diagnosis of cystic fibrosis of the pancreas as he did. The death of a sibling during early life from bronchopneumonia and the almost constant respiratory infection in the patient along with digestive disturbances certainly would make one think of this disease; however, I do not recall that any of our patients who have had cystic fibrosis of the pancreas ever vomited except one who had pyloric stenosis. After this was corrected surgically the vomiting ceased and the characteristic stools then became evident. This patient did not have cystic fibrosis of the pancreas. Necropsy disclosed an unusual finding associated with the esophagus.

At autopsy this baby boy, aged eight months, appeared to be two months younger than the stated age. The body was poorly nourished and weighed 6.25 kg. A recently made incision above the left sterno-clavicular articulation was found. The findings of interest were all located in the neck and thorax.

On dissecting out the structures of the neck a diverticulum of the esophagus was found coming off from the posterior wall between the inferior constrictor muscle and the crico-pharyngeus muscle. The diverticulum extended posteriorly for a distance of 4.5 cm. where it entered the apex of the right lung which was enveloped by dense adhesions.

The upper half of the upper lobe of the right lung was firm and gray. When sectioned it was found to be the site of an abscess which contained creamy greenish-brown pus. The wall of the abscess measured 8 mm. The remainder of the lung was not remarkable except for a few consolidated areas in the lower lobe. The bronchial lymph nodes on the right were enlarged. The left lung was partly atelectatic. The gross findings in the other organs were not unusual, the liver being congested.

Microscopically, the lung and pleura showed evidence of subacute and chronic inflammation with considerable production of connective tissue. In the heart, one of the arteries showed evidence of medial degeneration with deposition of calcium, an infiltration of polymorphonuclear leukocytes and round cells being noted about the vessel. In the adrenals some deposition of calcium in a blood vessel wall was observed and one vessel has been thrombosed and recanalized. The kidneys exhibit thickening of the base-

ment membrane of some of the glomerular tufts, occasional hyalinization and thickening of Bowman's capsule being seen. The convoluted tubules show swelling.

PATHOLOGICAL DIAGNOSIS

Abscess of right lung, upper lobe secondary to
Rupture of congenital esophageal diverticulum
Adhesive pleuritis, right
Bronchopneumonia, right lung, lower lobe
Atelectasis, left lung.
Vascular degenerative changes involving arteries of the heart and adrenals.
Thrombosis of adrenal arteriole
Subacute diffuse glomerulonephritis.

These findings have been unusual in our experience and to my knowledge this is the only infant who has exhibited this abnormality of the esophagus during the past twenty-five years at this hospital. Jackson and Jackson[1] state that an actual pouch at birth is a rare finding but a congenital weak point is not. They state that the usual classification of pulsion, traction and congenital are inaccurate as two or three factors may enter into the causation of all diverticula of the esophagus and hypopharynx.

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